* FILE 'USPAT' ENTERED AT 15:22:16 ON 14 DEC 95

* WELCOME TO TH
E * * U.S. PATENT TEXT FILE * * * * * * * * * * * * * *
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=> s (cancer or tumor)(p)(irradiat?)
14468 CANCER
11519 TUMOR
62280 IRRADIAT?
L1 984 (CANCER OR TUMOR)(P)(IRRADIAT?)

=> s l1 and melanoma
2138 MELANOMA
L2 124 L1 AND MELANOMA

=> d l2 1-30

1. 5,475,092, Dec. 12, 1995, Cell binding agent conjugates of analogues and derivatives of CC-1065; Ravi V. J. Chari, et al., 530/391.7; 424/178.1, 179.1, 181.1; 530/391.9; 548/420, 427, 430, 433 [IMAGE AVAILABLE]

2. 5,473,070, Dec. 5, 1995, Substituted long chain alcohol xanthine compounds; Gail Underiner, et al., 544/267 [IMAGE AVAILABLE]

3. 5,470,730, Nov. 28, 1995, Method for producing T.sub.H -independent cytotoxic T lymphocytes; Phillip D. Greenberg, et al., 435/172.3; 424/93.21; 435/69.1, 69.52, 70.4, 252.3, 320.1 [IMAGE AVAILABLE]

4. 5,470,577, Nov. 28, 1995, Stimulation of tanning by DNA fragments or single-stranded DNA; Barbara A. Gilchrest, et al., 424/450, 59, 520, 561; 514/44 [IMAGE AVAILABLE]

5. 5,466,679, Nov. 14, 1995, Carboranyl uridines and their use in boron neutron capture therapy; Albert H. Soloway, et al., 514/50; 424/1.11; 514/64; 536/28.53 [IMAGE AVAILABLE]

6. 5,464,833, Nov. 7, 1995, Apoptosis regulating composition; Satoru Nakai, et al., 514/251, 249, 250, 255 [IMAGE AVAILABLE]

7. 5,464,436, Nov. 7, 1995, Method of performing laser therapy; Chadwick F. Smith, 607/89; 606/3, 9, 13 [IMAGE AVAILABLE]

8. 5,462,724, Oct. 31, 1995, Sensitizing agents for use in boron neutron capture therapy; Raymond F. Schinazi, et al., 424/1.77; 514/64; 558/72 [IMAGE AVAILABLE]

9. 5,455,022, Oct. 3, 1995, Halogenated sulfidohydroboranes for nuclear medicine and boron neutron capture therapy; Michiko Miura, et al., 424/1.61; 423/276 [IMAGE AVAILABLE]

10. 5,446,157, Aug. 29, 1995, Boron difluoride compounds useful in photodynamic therapy and production of laser light; Lee R. Morgan, et al., 546/13; 548/110, 405 [IMAGE AVAILABLE]

11. 5,445,608, Aug. 29, 1995, Method and apparatus for providing light-activated therapy; James C. Chen, et al., 604/20, 19, 21 [IMAGE AVAILABLE]

12. 5,440,041, Aug. 8, 1995, Acetal or ketal substituted xanthine compounds; Alistair Leigh, et al., 544/267, 268 [IMAGE AVAILABLE]

13. 5,439,942, Aug. 8, 1995, Method of treating certain tumors using illudin analogs; Michael J. Kelner, et

al., 514/691 [IMAGE AVAILABLE]
14. 5,439,936, Aug. 8, 1995, Method of treating certain tumors using illudin analogs; Michael J. Kelner, et al., 514/546, 678, 681, 691 [IMAGE AVAILABLE]

15. 5,434,076, Jul. 18, 1995, Tumor-specific, cell surface-binding monoclonal antibodies; Ralph S. Freedman, et al., 435/240.27, 70.21, 172.2; 530/388.15, 388.8, 388.85, 389.7, 865 [IMAGE AVAILABLE]
16. 5,424,296, Jun. 13, 1995, 2-Halo-2'-deoxyadenosines as therapeutic agents against malignant astrocytoma; Alan Saven, et al., 514/46; 536/27.63, 27.7 [IMAGE AVAILABLE]

17. 5,416,064, May 16, 1995, Cytotoxic agents comprising maytansinoids and their therapeutic use; Ravi J. Chari, et al., 514/229.5; 540/462 [IMAGE AVAILABLE]

18. 5,411,884, May 2, 1995, Monoclonal antibody L53 which recognizes a human tumor-associated antigen; Ingegerd Hellstrom, et al., 435/240.27, 70.21, 172.2; 530/387.3, 387.9, 388.15, 388.3, 391.3 [IMAGE AVAILABLE]
19. 5,407,925, Apr. 18, 1995, Regional chemotherapy within the central nervous system with 4-hydroperoxycyclophosphamide; Darell D. Bigner, et al., 514/110 [IMAGE AVAILABLE]

20. 5,405,598, Apr. 11, 1995, Sensitizing agents for use in boron neutron capture therapy; Raymond F. Schinazi, et al., 424/1.81, 1.77, 9.1; 514/64; 544/229, 238; 549/333; 556/436; 562/7, 20; 568/6, 328, 381 [IMAGE AVAILABLE]

21. 5,399,583, Mar. 21, 1995, Method of treating skin diseases; Julia G. Levy, et al., 514/410, 2, 185 [IMAGE AVAILABLE]

22. 5,395,924, Mar. 7, 1995, Blocked lectins; methods and affinity support for making the same using affinity ligands; and method of killing selected cell populations having reduced non-selective cytotoxicity; Walter A. Blattler, et al., 530/396; 424/178.1, 182.1; 530/370, 389.2, 391.7, 402, 408, 409 [IMAGE AVAILABLE]

23. 5,392,319, Feb. 21, 1995, Accelerator-based neutron irradiation; Philip E. Eggers, 376/194, 151 [IMAGE AVAILABLE]

24. 5,382,521, Jan. 17, 1995, Method of determining metastatic potential of bladder tumor cells; Avraham Raz, et al., 435/7.23; 436/64, 813; 530/387.7, 388.8, 388.85 [IMAGE AVAILABLE]

25. 5,376,800, Dec. 27, 1994, High resolution track etch autoradiography; Guido Solares, et al., 250/472.1 [IMAGE AVAILABLE]
26. 5,370,868, Dec. 6, 1994, Therapeutic use of vitaletheine modulators in neoplasia; Galen D. Knight, et al., 424/78.08, 78.37; 514/563 [IMAGE AVAILABLE]

27. 5,364,619, Nov. 15, 1994, Oncoimmunins; Beverly Packard, et al., 424/85.1, 85.2; 514/2; 530/350, 351 [IMAGE AVAILABLE]

28. 5,354,756, Oct. 11, 1994, Olefin-substituted long chain xanthine compounds; Gail Underiner, et al., 514/263; 544/267, 272, 273 [IMAGE AVAILABLE]

29. 5,354,686, Oct. 11, 1994, Extracellular matrix protein adherent T cells; Allan B. Haberman, 435/240.2, 240.23, 240.243 [IMAGE AVAILABLE]
30. 5,341,292, Aug. 23, 1994, Monte Carlo based treatment planning for neutron capture therapy; Robert G. Zamenhof, 364/413.13, 413.26 [IMAGE AVAILABLE]

=> d l2 3, 21 cit ab

3. 5,470,730, Nov. 28, 1995, Method for producing T.sub.H -independent cytotoxic T lymphocytes; Phillip D.

Greenberg, et al., 435/172.3; 424/93.21; 435/69.1, 69.52, 70.4, 252.3, 320.1 [IMAGE AVAILABLE]
US PAT NO: 5,470,730 [IMAGE AVAILABLE] L2: 3 of 124
ABSTRACT:
The present invention relates to cytotoxic T lymphocytes (CTLs) which have been converted to a helper T cell independent phenotype by introducing a recombinant expression vector encoding IL-1 receptor into the CTL. The resulting CTLs have the ability to grow or function independent of T cell help.

21. 5,399,583, Mar. 21, 1995, Method of treating skin diseases; Julia G. Levy, et al., 514/410, 2, 185 [IMAGE AVAILABLE]

US PAT NO: 5,399,583 [IMAGE AVAILABLE] L2: 21 of 124
ABSTRACT:
A group of hydro-monobenzoporphyrins "green porphyrins" (Gp) having absorption maxima in the range of 670-780 nanometers is useful in treating disorders or conditions which are subject to hematoporphyrin derivative (HPD) treatment in the presence of light, or in treating virus, cells and tissues generally to destroy unwanted targets. The use of the Gp of the invention permits the irradiation for therapy to use wavelengths other than those absorbed by blood. The Gp of the invention may also be conjugated to ligands specific for receptor or to specific immunoglobulins or fragments thereof to target specific tissues or cells for the radiation treatment. Use of these materials permits lower levels of drug to be used, thus preventing side reactions which might destroy normal tissues.

=> s l2 and cyclophosphamide
1031 CYCLOPHOSPHAMIDE
L3 15 L2 AND CYCLOPHOSPHAMIDE

=> d l3 1-2

1. 5,407,925, Apr. 18, 1995, Regional chemotherapy within the central nervous system with 4-hydroperoxycyclophosphamide; Darell D. Bigner, et al., 514/110 [IMAGE AVAILABLE]

2. 5,395,924, Mar. 7, 1995, Blocked lectins; methods and affinity support for making the same using affinity ligands; and method of killing selected cell populations having reduced non-selective cytotoxicity; Walter A. Blattler, et al., 530/396; 424/178.1, 182.1; 530/370, 389.2, 391.7, 402, 408, 409 [IMAGE AVAILABLE]

=> s l1 and hapten
2013 HAPTEN
L4 21 L1 AND HAPTEN

=> d l4 1-5

1. 5,474,772, Dec. 12, 1995, Method of treatment with medical agents; Stephen W. Maddock, 424/140.1; 604/5, 6, 28 [IMAGE AVAILABLE]
2. 5,433,896, Jul. 18, 1995, Dibenzopyrromethencboron difluoride dyes; Hee C. Kang, et al., 252/700, 301.16, 301.34, 301.35; 548/110, 405 [IMAGE AVAILABLE]

3. 5,428,154, Jun. 27, 1995, Complexes of functionalized tetraazacyclododecane chelates with bismuth, lead, ytriium, actinium, or lanthanide metal ions; Otto A. Gansow, et al., 540/465; 534/10, 14, 15, 16; 540/452, 474 [IMAGE AVAILABLE]

4. 5,428,139, Jun. 27, 1995, Bicyclopolyazamacrocyclophosphonic acid complexes for use as radiopharmaceuticals; Garry E. Kiefer, et al., 534/10, 14 [IMAGE AVAILABLE]

5. 5,342,604, Aug. 30, 1994, Complexes possessing ortho ligating functionality; David A. Wilson, et al., 424/1.65; 534/10 [IMAGE AVAILABLE]

=> s irradiat?(p)tumor(P)hapten
62280 IRRADIAT?
11519 TUMOR
2013 HAPTEN
L5 4 IRRADIAT?(P)TUMOR(P)HAPTEN

=> d l6 1
'L6' NOT FOUND

=> d l5 1

1. 5,428,154, Jun. 27, 1995, Complexes of functionalized tetraazacyclododecane chelates with bismuth, lead, ytriium, actinium, or lanthanide metal ions; Otto A. Gansow, et al., 540/465; 534/10, 14, 15, 16; 540/452, 474 [IMAGE AVAILABLE]

=> d l4 1 cit ab

1. 5,474,772, Dec. 12, 1995, Method of treatment with medical agents; Stephen W. Maddock, 424/140.1; 604/5, 6, 28 [IMAGE AVAILABLE]
US PAT NO: 5,474,772 [IMAGE AVAILABLE] L4: 1 of 21
ABSTRACT:
A method of therapeutic or diagnostic treatment using a medical agent in which there will be beneficial effects if the level of circulating medical agent is reduced artificially faster than the reduction by the normal clearance or routes that obviate normal clearance mechanisms. The method includes the extracorporeal removal of the medical agent by passing bodily fluid from the patient over a support adapted to selectively immobilize the medical agent.

=> s peptide? and melanoma/ti
17794 PEPTIDE?
29 MELANOMA/TI
L6 5 PEPTIDE? AND MELANOMA/TI

=> d l6 1-5

1. 5,331,014, Jul. 19, 1994, Effect of N,N,N-trimethylsphingosine on protein kinase-C activity; **melanoma** cell growth in vitro; metastatic potential in vivo and human platelet aggregation; Satoshi Kimura, et al., 514/642; 435/240.1, 240.2 [IMAGE AVAILABLE]

2. 5,270,202, Dec. 14, 1993, Anti-idiotypic antibodies to human **melanoma**-associated proteoglycan antigen; Syamal Raychaudhuri, 435/240.27; 530/387.2, 387.3, 388.8, 388.85, 389.7 [IMAGE AVAILABLE]
3. 5,262,177, Nov. 16, 1993, Recombinant viruses encoding the human **melanoma**-associated antigen; Joseph P. Brown, et al., 435/235.1; 424/185.1, 199.1, 232.1; 435/69.3, 172.3, 240.2, 252.3, 252.33, 320.1; 530/350; 536/23.5; 935/9, 32, 41, 57, 65, 70, 73 [IMAGE AVAILABLE]
4. 5,141,742, Aug. 25, 1992, Vaccines against **melanoma**; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

5. 4,849,509, Jul. 18, 1989, Monoclonal antibodies against **melanoma**-associated antigens and hybrid cell lines producing these antibodies; Jan Thurin, et al., 530/388.2; 424/137.1, 156.1; 435/70.21, 172.2, 172.3, 240.27; 530/388.85, 806, 828, 861 [IMAGE AVAILABLE]
=> d l4 4 cit ab

4. 5,428,139, Jun. 27, 1995, Bicyclopolyazamacrocyclophosphonic acid complexes for use as radiopharmaceuticals; Garry E. Kiefer, et al., 534/10, 14 [IMAGE AVAILABLE]

US PAT NO: 5,428,139 [IMAGE AVAILABLE] L4: 4 of 21
ABSTRACT:
Complexes of bicyclopolyazamacrocyclophosphonic acid compounds with a metal ion, e.g. .sup.153 Sm, .sup.177 Lu, .sup.159 Gd, .sup.149 Pm, .sup.140 La, .sup.175 Yb, .sup.166 Ho, .sup.90 Y, .sup.47 Sc, .sup.186 Re, .sup.188 Re, .sup.142 Pr, .sup.99m Tc, .sup.67 Ga, .sup.68 Ga, .sup.105 Rh, .sup.97 Ru, .sup.111 In, .sup.113m In or .sup.115m In ion, are disclosed. The complexes can be covalently attached to a biologically active molecule, e.g. an antibody or antibody fragment, to form conjugates. The complexes and conjugates are useful as
radiopharmaceutical agents for therapy and/or diagnostic purposes.
=> d l6 4 cit ab

4. 5,141,742, Aug. 25, 1992, Vaccines against **melanoma**; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

US PAT NO: 5,141,742 [IMAGE AVAILABLE] L6: 4 of 5
ABSTRACT:
Peptides or proteins related to a melanoma associated antigen are described. These are produced in large quantities via recombinant DNA techniques and/or by chemical synthetic methods. The **peptides** or proteins can be used as immunogens in vaccine formulations which can induce an immune response that selectively destroys melanoma cells in a vaccinated individual. Where the **peptides** or proteins are expressed by a recombinant virus, inactivated or live virus vaccine formulations may be prepared.

=> s 5141742/pn and hapten?
1 5141742/PN
2738 HAPTEN?
L7 1 5141742/PN AND HAPTEN?

=> d l7 kwic
US PAT NO: **5,141,742** [IMAGE AVAILABLE] L7: 1 of 1
SUMMARY:

BSUM(59)

Vaccine . . . altered, unaltered, modified or unmodified, will be referred to a "p97 related peptides". Where the p97 related peptide is a **hapten** (i.e., antigenic but not immunogenic) the **hapten** can be conjugated to a carrier molecule that confers immunogenicity.
DETDESC:

DETD(73)

In instances where the p97-related peptide is a **hapten**, i.e., a molecule that is antigenic in that it can react selectively with cognate antibodies, but not immunogenic in that it cannot elicit an immune response, the **hapten** may be covalently bound to a carrier or immunogenic molecule and the **hapten**-carrier may be formulated for use as a vaccine; for instance, a large protein such as protein serum albumin will confer immunogenicity to the **hapten** coupled to it.
=> s 5141742/pn and irradiat?
1 5141742/PN
62280 IRRADIAT?
L8 1 5141742/PN AND IRRADIAT?

=> d l8 1 kwic
US PAT NO: **5,141,742** [IMAGE AVAILABLE] L8: 1 of 1
SUMMARY:

BSUM(50)

Studies . . . cancer vaccines is disputed, in spite of some reports of success. In many cases the vaccine preparations have consisted of **irradiated** tumor cells or tumor cells killed by exposure to certain chemical agents. Because pure human tumor-associated antigens have not been. . .

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=> s 5141742/pn and cyclophosphamide
          1 5141742/PN
       1031 CYCLOPHOSPHAMIDE
L9        0 5141742/PN AND CYCLOPHOSPHAMIDE

=> s peptide and melanoma?/ab
      14219 PEPTIDE
         99 MELANOMA?/AB
L10      15 PEPTIDE AND MELANOMA?/AB

=> s peptide ?and melanoma?/ab

       1937 PEPTIDE/AB
     100044 ?AND/AB
         99 MELANOMA?/AB
L11      0 PEPTIDE ?AND MELANOMA?/AB
           ((PEPTIDE(W)?AND(W)MELANOMA?)/AB)

=> s peptide? and melanoma?/ab
      17794 PEPTIDE?
         99 MELANOMA?/AB
L12      20 PEPTIDE? AND MELANOMA?/AB

=> d l12 1-20
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1. 5,449,766, Sep. 12, 1995, DNA encoding NEI and NGE **peptides**; Joan Vaughan, et al., 536/23.5; 435/69.1, 69.4, 240.2, 252.3, 320.1; 530/300, 326; 536/22.1, 23.1 [IMAGE AVAILABLE]

2. 5,270,202, Dec. 14, 1993, Anti-idiotypic antibodies to human melanoma-associated proteoglycan antigen; Syamal Raychaudhuri, 435/240.27; 530/387.2, 387.3, 388.8, 388.85, 389.7 [IMAGE AVAILABLE]

3. 5,262,177, Nov. 16, 1993, Recombinant viruses encoding the human melanoma-associated antigen; Joseph P. Brown, et al., 435/235.1; 424/185.1, 199.1, 232.1; 435/69.3, 172.3, 240.2, 252.3, 252.33, 320.1; 530/350; 536/23.5; 935/9, 32, 41, 57, 65, 70, 73 [IMAGE AVAILABLE]

4. 5,252,718, Oct. 12, 1993, Fibroblast growth factor antagonists; J. Andrew Baird, et al., 530/399, 324, 325, 326, 327, 328; 930/21, 120, DIG.530 [IMAGE AVAILABLE]

5. 5,221,622, Jun. 22, 1993, 170kD membrane-bound protease useful in diagnosis of malignant cellular transformation; Wen-Tien Chen, 435/226, 219; 530/828 [IMAGE AVAILABLE]

6. 5,147,638, Sep. 15, 1992, Inhibition of tumor growth by blockade of the protein C system; Charles T. Esmon, et al., 424/85.1, 85.2, 85.4, 85.5, 152.1, 172.1, 282.1; 435/212; 514/2, 8, 12; 530/351, 381, 388.25, 389.3 [IMAGE AVAILABLE]

7. 5,145,677, Sep. 8, 1992, Process for treatment of diseases; Johann-Friedrich von Eichborn, et al., 424/85.5, 85.1, 85.2 [IMAGE AVAILABLE]

8. 5,141,742, Aug. 25, 1992, Vaccines against melanoma; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

9. 5,132,408, Jul. 21, 1992, Fibroblast growth factor antagonists; Andrew J. Baird, et al., 530/399, 324, 325, 326, 327, 330 [IMAGE AVAILABLE]

10. 5,132,214, Jul. 21, 1992, Large scale production of plasminogen activator from normal human colon cells; Joseph Feder, et al., 435/70.3; 424/94.63, 94.64; 435/212, 219; 514/8; 530/395 [IMAGE AVAILABLE]
11. 5,049,655, Sep. 17, 1991, Melanin-concentrating hormones; Joan Vaughan, et al., 530/326; 435/69.4, 320.1; 530/827, 854; 536/23.51 [IMAGE AVAILABLE]

12. 5,039,794, Aug. 13, 1991, Tumor egress factor and processes for producing the same; Marjorie L. Wier, et al., 530/399, 350, 351, 380, 395, 413, 414, 415, 416, 417 [IMAGE AVAILABLE]

13. 5,039,793, Aug. 13, 1991, Cytoplasmic protein of suprabasal epidermal cells, antibodies capable of recognizing said protein, and hybrid cellular stocks capable of secreting such antibodies; Bruno Bernard, et al., 530/350; 435/70.21, 240.27; 530/388.2, 389.1, 391.1; 935/104 [IMAGE AVAILABLE]

14. 4,877,611, Oct. 31, 1989, Vaccine containing tumor antigens and adjuvants; John L. Cantrell, 424/277.1, 278.1, 282.1; 514/885, 937, 938, 939, 943 [IMAGE AVAILABLE]

15. 4,851,517, Jul. 25, 1989, Tissue plasminogen activator oligosaccharide from normal human colon cells; Joseph Feder, et al., 536/53, 1.11, 123 [IMAGE AVAILABLE]

16. 4,816,442, Mar. 28, 1989, Method of inhibiting tumor growth sensitive to CIF-.beta.treatment; John M. McPherson, et al., 514/12, 2; 930/10, 120 [IMAGE AVAILABLE]

17. 4,786,593, Nov. 22, 1988, Diagnostic method for detection of neural crest disease; Alonzo Ross, et al., 435/7.23; 436/503, 504, 518, 548, 813, 817; 935/110 [IMAGE AVAILABLE]

18. 4,783,313, Nov. 8, 1988, Enhancing body defense mechanisms; Jack G. Makari, 424/1.57, 1.69, 277.1, 573; 514/2, 44, 88 [IMAGE AVAILABLE]
19. 4,751,084, Jun. 14, 1988, Tissue plasminogen activator from normal human colon cells; Joseph Feder, et al., 424/94.64, 94.63; 435/212, 219; 514/8, 54, 822; 530/395; 536/123 [IMAGE AVAILABLE]

20. 4,705,677, Nov. 10, 1987, Immunization; Jack G. Makari, 424/1.57, 277.1; 514/2, 8, 44 [IMAGE AVAILABLE]

=> d l12 20 cit ab

20. 4,705,677, Nov. 10, 1987, Immunization; Jack G. Makari, 424/1.57, 277.1; 514/2, 8, 44 [IMAGE AVAILABLE]

US PAT NO: 4,705,677 [IMAGE AVAILABLE] L12: 20 of 20
ABSTRACT:
Disclosed is a method of providing immuno-prophylaxis as well as malignant tumor immuno-therapy by administering a malignant-tumor- immunization and recession-provoking antigenic agent of malignant tumor tissue source to a living subject in an effective dosage and regimen to develop a sufficiently high level of defense against malignant tumor incipience, or to retard such tumor development and/or provoke its recession. The malignant-tumor-immunization and recession-provoking antigenic agent or tumor tissue source is a combination of:
(a) the tritiated form of glycoprotein polysaccharide-like-antigenic substance (TPS) obtained by removing lipids and protein from a differential centrifugation sediment of a saline suspension of the mitochondrial fraction of cancer tissue, e.g. from carcinoma, fibrosarcoma, lymphoma, or **melanoma**: and
(b) deoxyribonucleic acid (DNA) derived from the nuclear fraction of malignant tumor tissue; or

AT L1 E1 V1 X0 DT 92024290956

File 155:MEDLINE(R) 1966-1995/Nov W4
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Set Items Description
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?s (tumor? or cancer? or melanoma) and (vaccin? or immuniz?)
331668 TUMOR?
217829 CANCER?
32424 MELANOMA
75882 VACCIN?
66470 IMMUNIZ?
S1 9282 (TUMOR? OR CANCER? OR MELANOMA) AND (VACCIN? OR IMMUNIZ?) ?s s1 and peptide?

9282 S1
204335 PEPTIDE?
S2 419 S1 AND PEPTIDE?
?s (tumor? or cancer? or melanoma)/ti and (vaccin? or immuniz?)/ti
121389 TUMOR?/TI
149810 CANCER?/TI
15508 MELANOMA/TI
33976 VACCIN?/TI
12564 IMMUNIZ?/TI
S3 843 (TUMOR? OR CANCER? OR MELANOMA)/TI AND (VACCIN? OR IMMUNIZ?)/TI
?s s3 and peptide?

843 S3
204335 PEPTIDE?
S4 24 S3 AND PEPTIDE?
?t s4/6/1-24

?t s4/7/6,11,12,15

4/7/6
DIALOG(R)File 155:MEDLINE(R)

08887952 94202952
Human ***cancer*** ***vaccines***.
Sinkovics J; Horvath J; Szabo-Szabari M
Cancer Institute, St. Joseph's Hospital, Tampa, Florida 33607. Leukemia (ENGLAND) Apr 1994, 8 Suppl 1 pS194-7, ISSN 0887-6924 Journal Code: LEU
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL Immune T cells recognize ***peptide*** antigens presented to them within self-MHC molecules. Thus auto-tumor reactive lymphocyte populations can be generated. Antigenic expression can be modified and intensified and reactive lymphocyte populations can be expanded. Active immunization of the tumor-bearing human host can induce

immune reactions of tumor rejection strength. Frequently, micrometastases can be eliminated and occasionally partial or complete remissions of gross metastases can be induced. (22 Refs.)

4/7/11
DIALOG(R)File 155:MEDLINE(R)

08738839 94053839
Prospects of T-cell immunotherapy for ***cancer*** by ***peptide*** ***vaccination***.
Melief CJ
Department of Immunohaematology and Blood Bank, University Hospital Leiden, The Netherlands.
Semin Hematol (UNITED STATES) Jul 1993, 30 (3 Suppl 3) p32-3, ISSN 0037-1963 Journal Code: UN9
Languages: ENGLISH
Document type: JOURNAL ARTICLE

4/7/12
DIALOG(R)File 155:MEDLINE(R)

08694222 94009222
Immunization against ***tumor*** and minor histocompatibility antigens by eluted cellular ***peptides*** loaded on antigen processing defective cells.
Franksson L; Petersson M; Kiessling R; Karre K
Microbiology and Tumor Biology Center, Karolinska Institutet, Stockholm, Sweden.
Eur J Immunol (GERMANY) Oct 1993, 23 (10) p2606-13, ISSN 0014-2980 Journal Code: EN5
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Material eluted from RMA lymphoma or B6 spleen cells under acid conditions was fractionated by reverse phase high-performance liquid chromatography, and tested for ability to restore the sensitivity to cytotoxic T lymphocytes of the processing/presentation defective mutant line RMA-S. This allowed identification of three fractions (termed M1, M2 and M3) carrying B6 antigens recognized by cytotoxic T lymphocytes (CTL) elicited across the minor histocompatibility barrier A.BY anti-B6 (both H-2b) and one fraction (termed T1) carrying a tumor antigen recognized by B6 anti-RMA CTL. By parallel runs of material from cell lysates over major histocompatibility complex class I affinity columns, the M2 and M3 antigens were defined as Kb restricted, and M1 and T1 as Db restricted. Isolated fractions loaded onto RMA-S cells could be used to prime anti-minor histocompatibility antigen and tumor CTL in vivo. They could also be used for in vitro restimulation of spleen cells from mice that had been primed either by antigen-loaded RMA-S, or by wild-type RMA tumor cells and B6 splenocytes. The CTL generated by these methods were specific for the loading antigen, and they also recognized the antigen on the "physiological" target, i.e. RMA or B6 lymphoblasts. This system based on RMA-S as an immunization and target antigen reporter cell may be used for dissection of complex CTL responses, e.g. in studies of clonal composition and epitope dominance, or for studies of tumors that are poor stimulators of immunity.

4/7/15
DIALOG(R)File 155:MEDLINE(R)

08329802 93039802
Construction of ***cancer*** ***vaccines*** with carbohydrate and protein (***peptide***) ***tumor*** antigens.

Livingston PO
Department of Medicine, Memorial Sloan-Kettering Cancer Center, New York, New York 10021.
Curr Opin Immunol (ENGLAND) Oct 1992, 4 (5) p624-9, ISSN 0952-7915 Journal Code: AH1
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW LITERATURE Tumor vaccinology is as old as immunological thought and as young as our rapidly evolving understanding of antigen processing and presentation. The recent availability of carbohydrate and ***peptide*** tumor antigens suitable for vaccine construction, conjugate and recombinant vector technologies capable of augmenting helper and cytotoxic T cell activity and potent new immunological adjuvants have combined to produce considerable optimism for the future of tumor vaccines. (53 Refs.)
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$0.02 SPRNTNET
$0.14 Estimated cost this search
$5.79 Estimated total session cost 0.122 Hrs.

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-1995/Nov W4
(c) format only 1995 Knight-Ridder Info
File 5:BIOSIS PREVIEWS(R) 1969-1995/Oct W2
(c) 1995 BIOSIS
*File 5: s (Meeting()Abstract) or abstracts/DE for 1994+ conference records

Set Items Description
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?s (tumor? or cancer? or melanoma)/ti and (vaccin? or immuniz?)/ti
Processing
265489 TUMOR?/TI
277858 CANCER?/TI
31920 MELANOMA/TI
65079 VACCIN?/TI
26282 IMMUNIZ?/TI
S1 2171 (TUMOR? OR CANCER? OR MELANOMA)/TI AND (VACCIN? OR IMMUNIZ?)/TI
?s s1 and peptide?

2171 S1
440909 PEPTIDE?
S2 68 S1 AND PEPTIDE?
?rd

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...completed examining records

S3 51 RD (unique items)
?t s3/6/1-51

?t s3/7/26,30,33,34,38,41,46

3/7/26 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11800107 BIOSIS Number: 98400107
Vaccination with ***peptides*** encoding MuLV T helper and CTL epitopes protects for MuLV induced ***tumors***
Ossendorp F; Mengede E; Sijts A; Kast W M; Melief C J M
Bloodbank, Academic Hosp. Leiden, Leiden, Netherlands
0 (0). 1995. 879.
Full Journal Title: 9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY. The 9th International Congress of Immunology; Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies, San Francisco, California, USA, July 23-29, 1995. 311p. 9th International Congress of Immunology: San Francisco, California, USA. ISSN: *********
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 047 Iss. 009 Ref. 162510

3/7/30 (Item 6 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11732001 BIOSIS Number: 98332001
Polynucleotide ***vaccination*** for ***cancer*** treatment (Review) Spooner R A; Bel S J; Epenetos A A
Tumour Targeting Lab., ICRF Oncol. Unit, Dep. Clinical Oncol., Royal Postgraduate Med. Sch., MRC Build., Hammersmith Hosp., London W12 0HS, UK International Journal of Oncology 6 (6). 1995. 1203-1208. Full Journal Title: International Journal of Oncology
ISSN: 1019-6439
Language: ENGLISH
Print Number: Biological Abstracts Vol. 100 Iss. 003 Ref. 039725 Inoculation with pure naked DNA in the form of plasmids can stimulate both antibody and T-cell responses in vivo against plasmid-encoded proteins. ***Peptide*** products derived from cytosolic degradation of fragments of tumour-specific proteins, expressed de novo under the transcriptional control of strong mammalian or viral promoter/enhancer signals might gain access to the MHC Class I presentation pathway, mimicking the presentation of viral proteins in infected cells. Presentation as neo-antigens or surrogate antigens in this novel context may be a means of breaking immunological tolerance, and may lead to the generation of tumour-specific immune responses.

3/7/33 (Item 9 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11655458 BIOSIS Number: 98255458
Designing ***peptide*** ***vaccines*** for AIDS and ***cancer*** Berzofsky J A
Mol. Immunogenetics Vaccine Res. Sect., Metabolism Branch, Natl. Cancer Inst., NIH, Bethesda, MD 20892, USA
Cancer Biotherapy 10 (1). 1995. 78.

Full Journal Title: Second International Conference on Engineered Vaccines of Cancer and AIDS, San Francisco, California, USA, March 3-5, 1995. Cancer Biotherapy
ISSN: 1062-8401
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 047 Iss. 006 Ref. 102321

3/7/34 (Item 10 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11604566 BIOSIS Number: 98204566
Cellular immunity against DNA ***tumor*** viruses: Possibilities for ***peptide*** based ***vaccines*** and immune escapes
Toes R E M; Feltkamp M C W; Ressing M E; Offringa R; Grey H M; Sette A; Melief C J M; Kast W M
Dep. Immunohematol. and Blood Bank, University Hosp., P.O. Box 9600, 2300 RC Leiden, Netherlands
Journal of Cellular Biochemistry Supplement 0 (19A). 1995. 273. Full Journal Title: Keystone Symposium on Molecular Aspects of Viral Immunity, Keystone, Colorado, USA, January 16-23, 1995. Journal of Cellular Biochemistry Supplement
ISSN: 0733-1959
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 047 Iss. 005 Ref. 078229

3/7/38 (Item 14 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10951981 BIOSIS Number: 97151981
Use of synthetic ***peptides*** to ***immunize*** against breast ***cancer***
Apostolopoulos V; Xing P-X; Pietersz G A; McKenzie I F C Austin Res. Inst., Studley Road, Heidelberg 3084, VIC, AUL Journal of Leukocyte Biology 0 (SUPPL.). 1993. 88.
Full Journal Title: International Congress on the Regulation of Leukocyte Production and Immune Function held at the Joint Meeting of the Australasian Society for Immunology and Society for Leukocyte Biology, Sydney, New South Wales, Australia, December 1-5, 1993. Journal of Leukocyte Biology
ISSN: 0741-5400
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 004 Ref. 048582

3/7/41 (Item 17 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10215974 BIOSIS Number: 45015974
IMMUNIZATION AGAINST ***TUMOR*** AND MINOR HISTOCOMPATIBILITY ANTIGENS BY NATURALLY PROCESSED ***PEPTIDES***
FRANKSSON L; PETERSSON M; KIESSLING R; KARRE K
DEP. TUMOR BIOLOGY, KAROLINSKA INST., BOX 60 400, S-104 01 STOCKHOLM, SWEDEN.
KEYSTONE SYMPOSIUM ON CELLULAR IMMUNITY AND THE IMMUNOTHERAPY OF CANCER, TAOS, NEW MEXICO, USA, MARCH 17-24, 1993. J CELL BIOCHEM SUPPL 0 (17 PART D). 1993. 104. CODEN: JCBSD
Language: ENGLISH

3/7/46 (Item 22 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

(c) 1995 BIOSIS. All rts. reserv.

7912251 BIOSIS Number: 40113251
THE PRODUCTION OF SECOND GENERATION ANTI-***CANCER*** ANTIBODIES BY ***IMMUNIZATION*** WITH SYNTHETIC ***PEPTIDES***
MCKENZIE I F C; XING P-X
RESEARCH CENTER CANCER TRANSPLANTATION, DEP. PATHOLOGY, UNIVERSITY MELBOURNE, GRATTAN STREET, PARKVILLE, MELBOURNE, VICTORIA 3052, AUST. MEETING ON MONOCLONAL ANTIBODIES HELD AT THE 20TH ANNUAL MEETING OF THE KEYSTONE SYMPOSIA ON MOLECULAR AND CELLULAR BIOLOGY, DENVER, COLORADO, USA, MARCH 10-16, 1991. J CELL BIOCHEM SUPPL 15 (PART E). 1991. 130. CODEN: JCBSD
Language: ENGLISH

?s (tumor? or cancer? or melanoma) and (vaccin? or immuniz?) and peptide?
Processing
666876 TUMOR?
446490 CANCER?
62311 MELANOMA
137859 VACCIN?
111841 IMMUNIZ?
440909 PEPTIDE?
S4 863 (TUMOR? OR CANCER? OR MELANOMA) AND (VACCIN? OR IMMUNIZ?) AND PEPTIDE?

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>>>Unrecognizable Command

?s s4 and dt=review

863 S4
478442 DT=REVIEW
S5 50 S4 AND DT=REVIEW

?rd

...examined 50 records (50)
...completed examining records
S6 50 RD (unique items)

?t s6/6/1-50

?t s6/71,9,15,16,19,20,34,35,41

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>>>Format 41 is not valid in file 5

?t s6/7/1,9,15,16,19,20,34,35,41

6/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 1995 Knight-Ridder Info. All rts. reserv.

09402021 95332021
Strategies for gene therapy of ***melanoma***]
Strategien zur Gentherapie des Melanoms.

Dummer R; Davis-Daneshfar A; Dohring C; Dobbeling U; Burg G Dermatologische Klinik, Universitatsspital, Zurich.
Hautarzt (GERMANY) May 1995, 46 (5) p305-8, ISSN 0017-8470 Journal Code: G13
Languages: GERMAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL English Abstract
Active unspecific immunotherapy in an adjuvant or palliative setting has been shown to enhance survival in ***melanoma*** patients, and gene therapy now offers new perspectives for active specific immunotherapy. Gene therapy includes the transfer of genetic material performed by either viral or non-viral methods and in vivo or ex vivo. For ***melanoma*** the following approaches are suggested: ***vaccination*** with tumour-specific, HLA-associated antigens using ***peptides*** or 'naked DNA', ***vaccination*** with ***melanoma*** cells transfected with cytokine genes or B7, adoptive immunotherapy with specific T-lymphocytes or transfected tumour-infiltrating lymphocytes, or transfection of tumour cells with a tumour suppressor gene whose dysfunction plays a crucial role in ***melanoma***. (32 Refs.)

6/7/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09175072 95105072
T-cell adjuvants.
Hadden JW
Department of Internal Medicine, University of South Florida Medical College, Tampa 33612.
Int J Immunopharmacol (ENGLAND) Sep 1994, 16 (9) p703-10, ISSN 0192-0561 Journal Code: GRI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL T-cell adjuvancy involves the use of agents to stimulate preferentially delayed type hypersensitivity (DTH). Traditional adjuvants like Alum, Freunds, muramyl ***peptides***, and endotoxins are not selective. Natural infection (e.g. ***vaccinia***) may yield selective DTH. Low dose cyclophosphamide (CY) with mycobacteria was the first experimental T-cell adjuvant. New adjuvant formulations (ISCOMS, MAPS, etc.) with synthetic T-cell epitopes offer improved formulations. Upregulation of TH-1 helper cells and their actions with interleukins like IL-2, IL-12, and gamma IFN or antibodies to IL-4 and IL-10 may augment potently pathogen and ***tumor*** resistance. Similarly, transfection of ***tumor*** target cells with genes for IL-2, IL-12, gamma IFN, etc., offers novel ***vaccine*** treatment approaches. Finally, "thymomimetic" ***peptides*** like thymosin alpha 1 or drugs like levamisole or isoprinosine alone or in conjunction with interleukins may augment TH-1 and DTH responses. These approaches are seeing increasing emphasis in new treatment strategies for ***cancer*** and infections like HIV. (81 Refs.)

6/7/15 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08957887 94272887
New possibilities for ***cancer*** therapy with advances in ***cancer*** immunology.
MacLean GD; Longenecker BM
Department of Oncology, Faculty of Medicine, University of Alberta, Edmonton.
Can J Oncol (CANADA) Apr 1994, 4 (2) p249-54, ISSN 1183-2509 Journal Code: B01
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL There has been progress over the last decade in addressing three questions: Are there ***cancer***-associated antigens that could be targets for immunotherapy? Can the human immune system recognize ***cancer*** -associated

antigens? Can an anti-***cancer*** immune response affect ***cancer*** cells and lead to increased survival? Results from animal model studies have been interpreted by optimists as encouraging, and by pessimists as being irrelevant to human ***cancer***. Earlier studies on " ***cancer*** ***vaccines*** " utilized heterogeneous cell extracts of cell components. Monoclonal antibodies have enabled identification of relevant ***cancer*** -associated antigens or epitopes, such as the ganglioside GM2, the carbohydrates TF and STn, and the ***peptide*** sequences of MUC-1. In parallel with research on immune adjuvants and measures designed to inhibit suppressor activity, these epitopes are being tested for their potential in the immunotherapy of solid ***tumors***. It is clear that some of these ***cancer***-associated epitopes are immunogenic in humans. Mixed responses may relate to ***cancer*** heterogeneity and may indicate the importance of multi-epitopic ***vaccines*** . Responses are encouraging, but are they relevant? Prolonged disease stability challenges us to re-think the goals of ***cancer*** therapy. Recent advances in the knowledge of the effect of cytokines on ***tumor*** antigen expression and the regulation of the immune response, coupled with advances in active specific immunotherapy, provide hope that biomodulation may become an important part of the therapy of solid ***tumors*** in the next century. (32 Refs.)

6/7/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08887952 94202952
Human ***cancer*** ***vaccines***.
Sinkovics J; Horvath J; Szabo-Szabari M
Cancer Institute, St. Joseph's Hospital, Tampa, Florida 33607. Leukemia (ENGLAND) Apr 1994, 8 Suppl 1 pS194-7, ISSN 0887-6924 Journal Code: LEU
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Immune T cells recognize ***peptide*** antigens presented to them within self-MHC molecules. Thus auto-***tumor*** reactive lymphocyte populations can be generated. Antigenic expression can be modified and intensified and reactive lymphocyte populations can be expanded. Active ***immunization*** of the ***tumor***-bearing human host can induce immune reactions of ***tumor*** rejection strength. Frequently, micrometastases can be eliminated and occasionally partial or complete remissions of gross metastases can be induced. (22 Refs.)

6/7/19 (Item 19 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08792858 94107858
T-cell recognition of human ***melanoma*** antigens.
Kawakami Y; Nishimura MI; Restifo NP; Topalian SL; O'Neil BH; Shilyansky J; Yannelli JR; Rosenberg SA
Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892.
J Immunother (UNITED STATES) Aug 1993, 14 (2) p88-93, ISSN 1053-8550 Journal Code: AZ0
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The adoptive transfer of ***tumor***-infiltrating lymphocytes (TILs) with interleukin-2 (IL-2) has antitumor activity in some patients with metastatic ***melanoma*** . We have analyzed molecular mechanisms of TIL recognition of human ***melanoma***. Some cultured TILs specifically lysed autologous and some allogeneic melanomas sharing a variety of class I major histocompatibility complex (MHC) molecules. HLA-A2-restricted ***melanoma***-specific TILs lysed many HLA-A2+ ***melanoma*** cell lines from different patients but failed to lyse HLA-A2- ***melanoma*** and HLA-A2+ nonmelanoma cell lines. However, these TILs were capable of lysing many naturally HLA-A2- melanomas after introduction of the HLA-A2.1 gene by

vaccinia virus. These results indicate that shared ***melanoma*** antigens (Ag) are expressed in melanomas regardless of their human leukocyte antigen types. In order to identify these shared ***melanoma*** Ags, we have tested some known proteins expressed in ***melanoma***. Expression of tyrosinase or HMB45 Ag correlated with lysis of TILs. We are also attempting to isolate antigenic ***peptides*** by high performance liquid chromatography separation and genes encoding ***melanoma*** Ag by cDNA expression cloning. The T-cell component of the antimelanoma response was also analyzed by determining the genetic structure of the T-cell receptor (TCR) used by ***melanoma*** TILs. However, we did not observe common TCR variable region usage by different ***melanoma*** TILs. We could establish ***melanoma*** cell clones and lines resistant to TIL lysis due to the absence of or defects in the expression of Ag, MHC, or beta 2-microglobulin molecules. These data indicate multiple mechanisms for ***melanoma*** escape from T-cell immunosurveillance.(ABSTRACT TRUNCATED AT 250 WORDS) (20 Refs.)

6/7/20 (Item 20 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08744432 94059432
New strategies for enhancing the immunogenicity of ***tumors***. Pardoll DM
Johns Hopkins University School of Medicine, Baltimore.
Curr Opin Immunol (ENGLAND) Oct 1993, 5 (5) p719-25, ISSN 0952-7915 Journal Code: AH1
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL When a ***cancer*** grows in an individual, the immune system has either failed to recognize its antigens or failed to effectively respond. Increasing evidence for the existence of ***tumor*** antigens that are recognized by T cells provides a direct rationale for the design of novel strategies to either enhance ***tumor*** immunogenicity by genetic modification or utilize recombinant or ***peptide*** ***vaccines*** in cases where the relevant ***tumor*** antigens have been specifically identified. (33 Refs.)

6/7/34 (Item 34 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08150613 92288613
Synthetic ***peptides*** as ***vaccines***.
Rothbard JB
Biotechnology (UNITED STATES) 1992, 20 p451-65, ISSN 0740-7378 Journal Code: BIT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The economics of ***vaccines*** has been a major limitation in the commercial research and development of new approaches. This coupled with the natural scientific desire to simplify and define the composition of effective ***vaccines*** argues that the future of ***vaccines*** lies in novel approaches that will discover effective and less expensive components. ***Peptides*** , whether they are chemically synthesized or produced in bacteria, are an attractive possibility. To substitute linear ***peptides*** for complex mixtures of proteins would be a major technical advance and would stimulate tremendous commercial interest. However, at the present time I view this approach still unlikely to be of major practical importance. I conclude this because of the complexity of immunological responses to microorganisms. Even though, in some instances, a cytotoxic T-cell response or even the majority of the antibody response to a pathogen can be defined by a short linear ***peptide***, most people believe that multiple effector functions of the immune system should be stimulated in optimal ***vaccines***. For a small cocktail of ***peptides*** to reproduce the diversity of responses elicited by a virus, parasite, or bacterium is unlikely. However, I fully realize that remarkable progress has occurred towards understanding the structural requirements necessary to stimulate cellular and

humoral immune responses, and ***peptides*** have been integral in the development of this field. Also, the success of several research groups in developing effective antiviral ***vaccines*** using short linear ***peptides*** argues that I might be painting too dark of a picture. As someone who has used this strategy to explore ***peptide***-MHC and ***peptide***-antibody interactions, I am a strong scientific supporter of the approach. In this forum I am purposely cautious in my optimism. As the details of the complex molecular and cellular interactions that control the immune system are elucidated, both the number of strategies and the possible applications of modulating the immune response will increase as well. In addition to protective immunity to pathogens, ***cancer*** therapy could be revolutionized if ***tumor***-specific cytotoxic T-cells could be generated routinely. Novel therapeutic approaches to allergy, autoimmunity, and transplantation can be envisioned if the T-lymphocytes responsible for these syndromes could be modulated without total immune suppression. Consequently, I am confident that the experiments described in this chapter will be central to developing exciting new therapeutic and prophylactic compounds, but I am not sure that they will resemble naturally occurring ***peptides*** . The one aspect I am confident of is that the capacity of the immune response to protect the organism will continually surprise us. (40 Refs.)

6/7/35 (Item 35 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08135156 92273156
Tumor antigens.
Urban JL; Schreiber H
Department of Pathology, University of Chicago, Illinois 60637. Annu Rev Immunol (UNITED STATES) 1992, 10 p617-44, ISSN 0732-0582 Journal Code: ALO
Contract/Grant No.: CA-37156, CA, NCI; CA-22677, CA, NCI; CA-19266, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, ACADEMIC This review solidifies a new concept that common and rare types of human ***cancers*** harbor a variety of ***tumor***-specific mutant proteins that may be recognized as ***tumor***-specific antigens. These mutant proteins are encoded by oncogenes or suppressor genes that have undergone structural mutations resulting from point mutations, chromosomal translocations, internal deletions and viral insertional mutagenesis; several of these changes result in fusion proteins. While there is no evidence that immunosurveillance against these mutant proteins can prevent the development of primary ***cancers*** without prior ***immunization*** of the host, such ***tumor*** -specific molecules might be important for diagnosis and as targets for specific immunotherapy once the ***cancer*** has developed or even as targets for preventive ***cancer*** ***vaccines*** . Evidence further supports the notion that cytolytic or helper T cells are exquisitely selective in recognizing intracellular mutant proteins, and ***tumor***-specific T cell clones presently available may become useful for identifying previously unrecognized ***tumor*** -specific mutations. Many ***tumor***-specific mutant proteins clearly play a causative role in the establishment of malignant behavior, whereas other carcinogen-induced changes have at least immunological relevance. In any case strong evidence in mouse and man indicates that a single malignant cell can express multiple independent antigenic target sites. Such multiplicity may allow a multi-pronged immune attack that substantially decreases the chance of ***tumor*** escape. Future work must explore whether immune responses to ***tumor***-specific mutant proteins can lead to immunological ***tumor*** rejection and explore the possibility of chemically engineering ***tumor*** mutant ***peptides*** to be highly immunogenic, even in hosts that have previously failed to respond to the ***tumor***. (162 Refs.)

6/7/41 (Item 41 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07956170 92094170

T-cell immunotherapy of ***cancer***.

Melief CJ; Kast WM

Division of Immunology, The Netherlands Cancer Institute, Amsterdam. Res Immunol (FRANCE) Jun-Aug 1991, 142 (5-6) p425-9, ISSN 0923-2494 Journal Code: R6E

Languages: ENGLISH

Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL In animal systems, complete and permanent eradication of tumours can be achieved by adoptive transfer of MHC-restricted T cells, combined with IL2. In certain types of human ***cancer*** (***melanoma*** and perhaps renal cell carcinoma), tumour-specific T cells are probably the therapeutically most active cells among LAK or TIL cells. To prove these points, it is necessary to conduct trials with cloned tumour-specific T cells. Other potentially immunogenic ***tumors*** are cervical carcinoma, associated with human papilloma virus, and Burkitt's lymphoma, associated with Epstein-Barr virus. Most other human tumours, caused by subtle mutations in proto-oncogenes, are likely to be poorly or non-immunogenic. It is worthwhile trying to overcome this by ***vaccination*** with IL2 or IFN gamma-producing tumour cells or by deliberate ***vaccination*** with desirable targets for tumour-specific CTL such as the products of point-mutated oncogenes, including ras (Jung and Schleusener, 1991) and p53 (Rodriguez et al., 1990; Halevy et al., 1990), provided the relevant ***peptides*** are processed and bound to MHC class I molecules. Other potential targets are breakpoint ***peptides*** of translocated oncogene products such as bcr/abl (Van Denderen et al., 1990). In viral systems, it has already been established that ***peptide*** ***vaccination*** for protective CTL induction is feasible (Aichele et al., 1989; Schulz et al., 1991; Kast et al., 1991). (46 Refs.)

?logoff hold

Dummer R; Davis-Daneshfar A; Dohring C; Dobbeling U; Burg G Dermatologische Klinik, Universitatsspital, Zurich.
Hautarzt (GERMANY) May 1995, 46 (5) p305-8, ISSN 0017-8470 Journal Code: G13
Languages: GERMAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL English Abstract
Active unspecific immunotherapy in an adjuvant or palliative setting has been shown to enhance survival in ***melanoma*** patients, and gene therapy now offers new perspectives for active specific immunotherapy. Gene therapy includes the transfer of genetic material performed by either viral or non-viral methods and in vivo or ex vivo. For ***melanoma*** the following approaches are suggested: ***vaccination*** with tumour-specific, HLA-associated antigens using ***peptides*** or 'naked DNA', ***vaccination*** with ***melanoma*** cells transfected with cytokine genes or B7, adoptive immunotherapy with specific T-lymphocytes or transfected tumour-infiltrating lymphocytes, or transfection of tumour cells with a tumour suppressor gene whose dysfunction plays a crucial role in ***melanoma***. (32 Refs.)

6/7/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09175072 95105072
T-cell adjuvants.
Hadden JW
Department of Internal Medicine, University of South Florida Medical College, Tampa 33612.
Int J Immunopharmacol (ENGLAND) Sep 1994, 16 (9) p703-10, ISSN 0192-0561 Journal Code: GRI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL T-cell adjuvancy involves the use of agents to stimulate preferentially delayed type hypersensitivity (DTH). Traditional adjuvants like Alum, Freunds, muramyl ***peptides***, and endotoxins are not selective. Natural infection (e.g. ***vaccinia***) may yield selective DTH. Low dose cyclophosphamide (CY) with mycobacteria was the first experimental T-cell adjuvant. New adjuvant formulations (ISCOMS, MAPS, etc.) with synthetic T-cell epitopes offer improved formulations. Upregulation of TH-1 helper cells and their actions with interleukins like IL-2, IL-12, and gamma IFN or antibodies to IL-4 and IL-10 may augment potently pathogen and ***tumor*** resistance. Similarly, transfection of ***tumor*** target cells with genes for IL-2, IL-12, gamma IFN, etc., offers novel ***vaccine*** treatment approaches. Finally, "thymomimetic" ***peptides*** like thymosin alpha 1 or drugs like levamisole or isoprinosine alone or in conjunction with interleukins may augment TH-1 and DTH responses. These approaches are seeing increasing emphasis in new treatment strategies for ***cancer*** and infections like HIV. (81 Refs.)

6/7/15 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08957887 94272887
New possibilities for ***cancer*** therapy with advances in ***cancer*** immunology.
MacLean GD; Longenecker BM
Department of Oncology, Faculty of Medicine, University of Alberta, Edmonton.
Can J Oncol (CANADA) Apr 1994, 4 (2) p249-54, ISSN 1183-2509 Journal Code: B01
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL There has been progress over the last decade in addressing three questions: Are there ***cancer***-associated antigens that could be targets for immunotherapy? Can the human immune system recognize ***cancer*** -associated

antigens? Can an anti-***cancer*** immune response affect ***cancer*** cells and lead to increased survival? Results from animal model studies have been interpreted by optimists as encouraging, and by pessimists as being irrelevant to human ***cancer***. Earlier studies on " ***cancer*** ***vaccines*** " utilized heterogeneous cell extracts of cell components. Monoclonal antibodies have enabled identification of relevant ***cancer*** -associated antigens or epitopes, such as the ganglioside GM2, the carbohydrates TF and STn, and the ***peptide*** sequences of MUC-1. In parallel with research on immune adjuvants and measures designed to inhibit suppressor activity, these epitopes are being tested for their potential in the immunotherapy of solid ***tumors***. It is clear that some of these ***cancer***-associated epitopes are immunogenic in humans. Mixed responses may relate to ***cancer*** heterogeneity and may indicate the importance of multi-epitopic ***vaccines*** . Responses are encouraging, but are they relevant? Prolonged disease stability challenges us to re-think the goals of ***cancer*** therapy. Recent advances in the knowledge of the effect of cytokines on ***tumor*** antigen expression and the regulation of the immune response, coupled with advances in active specific immunotherapy, provide hope that biomodulation may become an important part of the therapy of solid ***tumors*** in the next century. (32 Refs.)

6/7/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08887952 94202952
Human ***cancer*** ***vaccines***.
Sinkovics J; Horvath J; Szabo-Szabari M
Cancer Institute, St. Joseph's Hospital, Tampa, Florida 33607. Leukemia (ENGLAND) Apr 1994, 8 Suppl 1 pS194-7, ISSN 0887-6924 Journal Code: LEU
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Immune T cells recognize ***peptide*** antigens presented to them within self-MHC molecules. Thus auto-***tumor*** reactive lymphocyte populations can be generated. Antigenic expression can be modified and intensified and reactive lymphocyte populations can be expanded. Active ***immunization*** of the ***tumor***-bearing human host can induce immune reactions of ***tumor*** rejection strength. Frequently, micrometastases can be eliminated and occasionally partial or complete remissions of gross metastases can be induced. (22 Refs.)

6/7/19 (Item 19 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08792858 94107858
T-cell recognition of human ***melanoma*** antigens.
Kawakami Y; Nishimura MI; Restifo NP; Topalian SL; O'Neil BH; Shilyansky J; Yannelli JR; Rosenberg SA
Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892.
J Immunother (UNITED STATES) Aug 1993, 14 (2) p88-93, ISSN 1053-8550 Journal Code: AZ0
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The adoptive transfer of ***tumor***-infiltrating lymphocytes (TILs) with interleukin-2 (IL-2) has antitumor activity in some patients with metastatic ***melanoma*** . We have analyzed molecular mechanisms of TIL recognition of human ***melanoma***. Some cultured TILs specifically lysed autologous and some allogeneic melanomas sharing a variety of class I major histocompatibility complex (MHC) molecules. HLA-A2-restricted ***melanoma***-specific TILs lysed many HLA-A2+ ***melanoma*** cell lines from different patients but failed to lyse HLA-A2- ***melanoma*** and HLA-A2+ nonmelanoma cell lines. However, these TILs were capable of lysing many naturally HLA-A2- melanomas after introduction of the HLA-A2.1 gene by

vaccinia virus. These results indicate that shared ***melanoma*** antigens (Ag) are expressed in melanomas regardless of their human leukocyte antigen types. In order to identify these shared ***melanoma*** Ags, we have tested some known proteins expressed in ***melanoma***. Expression of tyrosinase or HMB45 Ag correlated with lysis of TILs. We are also attempting to isolate antigenic ***peptides*** by high performance liquid chromatography separation and genes encoding ***melanoma*** Ag by cDNA expression cloning. The T-cell component of the antimelanoma response was also analyzed by determining the genetic structure of the T-cell receptor (TCR) used by ***melanoma*** TILs. However, we did not observe common TCR variable region usage by different ***melanoma*** TILs. We could establish ***melanoma*** cell clones and lines resistant to TIL lysis due to the absence of or defects in the expression of Ag, MHC, or beta 2-microglobulin molecules. These data indicate multiple mechanisms for ***melanoma*** escape from T-cell immunosurveillance.(ABSTRACT TRUNCATED AT 250 WORDS) (20 Refs.)

6/7/20 (Item 20 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08744432 94059432
New strategies for enhancing the immunogenicity of ***tumors***. Pardoll DM
Johns Hopkins University School of Medicine, Baltimore.
Curr Opin Immunol (ENGLAND) Oct 1993, 5 (5) p719-25, ISSN 0952-7915 Journal Code: AH1
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL When a ***cancer*** grows in an individual, the immune system has either failed to recognize its antigens or failed to effectively respond. Increasing evidence for the existence of ***tumor*** antigens that are recognized by T cells provides a direct rationale for the design of novel strategies to either enhance ***tumor*** immunogenicity by genetic modification or utilize recombinant or ***peptide*** ***vaccines*** in cases where the relevant ***tumor*** antigens have been specifically identified. (33 Refs.)

6/7/34 (Item 34 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08150613 92288613
Synthetic ***peptides*** as ***vaccines***.
Rothbard JB
Biotechnology (UNITED STATES) 1992, 20 p451-65, ISSN 0740-7378 Journal Code: BIT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The economics of ***vaccines*** has been a major limitation in the commercial research and development of new approaches. This coupled with the natural scientific desire to simplify and define the composition of effective ***vaccines*** argues that the future of ***vaccines*** lies in novel approaches that will discover effective and less expensive components. ***Peptides*** , whether they are chemically synthesized or produced in bacteria, are an attractive possibility. To substitute linear ***peptides*** for complex mixtures of proteins would be a major technical advance and would stimulate tremendous commercial interest. However, at the present time I view this approach still unlikely to be of major practical importance. I conclude this because of the complexity of immunological responses to microorganisms. Even though, in some instances, a cytotoxic T-cell response or even the majority of the antibody response to a pathogen can be defined by a short linear ***peptide***, most people believe that multiple effector functions of the immune system should be stimulated in optimal ***vaccines***. For a small cocktail of ***peptides*** to reproduce the diversity of responses elicited by a virus, parasite, or bacterium is unlikely. However, I fully realize that remarkable progress has occurred towards understanding the structural requirements necessary to stimulate cellular and

humoral immune responses, and ***peptides*** have been integral in the development of this field. Also, the success of several research groups in developing effective antiviral ***vaccines*** using short linear ***peptides*** argues that I might be painting too dark of a picture. As someone who has used this strategy to explore ***peptide***-MHC and ***peptide***-antibody interactions, I am a strong scientific supporter of the approach. In this forum I am purposely cautious in my optimism. As the details of the complex molecular and cellular interactions that control the immune system are elucidated, both the number of strategies and the possible applications of modulating the immune response will increase as well. In addition to protective immunity to pathogens, ***cancer*** therapy could be revolutionized if ***tumor***-specific cytotoxic T-cells could be generated routinely. Novel therapeutic approaches to allergy, autoimmunity, and transplantation can be envisioned if the T-lymphocytes responsible for these syndromes could be modulated without total immune suppression. Consequently, I am confident that the experiments described in this chapter will be central to developing exciting new therapeutic and prophylactic compounds, but I am not sure that they will resemble naturally occurring ***peptides*** . The one aspect I am confident of is that the capacity of the immune response to protect the organism will continually surprise us. (40 Refs.)

6/7/35 (Item 35 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08135156 92273156
Tumor antigens.
Urban JL; Schreiber H
Department of Pathology, University of Chicago, Illinois 60637. Annu Rev Immunol (UNITED STATES) 1992, 10 p617-44, ISSN 0732-0582 Journal Code: ALO
Contract/Grant No.: CA-37156, CA, NCI; CA-22677, CA, NCI; CA-19266, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, ACADEMIC This review solidifies a new concept that common and rare types of human ***cancers*** harbor a variety of ***tumor***-specific mutant proteins that may be recognized as ***tumor***-specific antigens. These mutant proteins are encoded by oncogenes or suppressor genes that have undergone structural mutations resulting from point mutations, chromosomal translocations, internal deletions and viral insertional mutagenesis; several of these changes result in fusion proteins. While there is no evidence that immunosurveillance against these mutant proteins can prevent the development of primary ***cancers*** without prior ***immunization*** of the host, such ***tumor*** -specific molecules might be important for diagnosis and as targets for specific immunotherapy once the ***cancer*** has developed or even as targets for preventive ***cancer*** ***vaccines*** . Evidence further supports the notion that cytolytic or helper T cells are exquisitely selective in recognizing intracellular mutant proteins, and ***tumor***-specific T cell clones presently available may become useful for identifying previously unrecognized ***tumor*** -specific mutations. Many ***tumor***-specific mutant proteins clearly play a causative role in the establishment of malignant behavior, whereas other carcinogen-induced changes have at least immunological relevance. In any case strong evidence in mouse and man indicates that a single malignant cell can express multiple independent antigenic target sites. Such multiplicity may allow a multi-pronged immune attack that substantially decreases the chance of ***tumor*** escape. Future work must explore whether immune responses to ***tumor***-specific mutant proteins can lead to immunological ***tumor*** rejection and explore the possibility of chemically engineering ***tumor*** mutant ***peptides*** to be highly immunogenic, even in hosts that have previously failed to respond to the ***tumor***. (162 Refs.)

6/7/41 (Item 41 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07956170 92094170

T-cell immunotherapy of ***cancer***.

Melief CJ; Kast WM

Division of Immunology, The Netherlands Cancer Institute, Amsterdam. Res Immunol (FRANCE) Jun-Aug 1991, 142 (5-6) p425-9, ISSN 0923-2494 Journal Code: R6E

Languages: ENGLISH

Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL In animal systems, complete and permanent eradication of tumours can be achieved by adoptive transfer of MHC-restricted T cells, combined with IL2. In certain types of human ***cancer*** (***melanoma*** and perhaps renal cell carcinoma), tumour-specific T cells are probably the therapeutically most active cells among LAK or TIL cells. To prove these points, it is necessary to conduct trials with cloned tumour-specific T cells. Other potentially immunogenic ***tumors*** are cervical carcinoma, associated with human papilloma virus, and Burkitt's lymphoma, associated with Epstein-Barr virus. Most other human tumours, caused by subtle mutations in proto-oncogenes, are likely to be poorly or non-immunogenic. It is worthwhile trying to overcome this by ***vaccination*** with IL2 or IFN gamma-producing tumour cells or by deliberate ***vaccination*** with desirable targets for tumour-specific CTL such as the products of point-mutated oncogenes, including ras (Jung and Schleusener, 1991) and p53 (Rodriguez et al., 1990; Halevy et al., 1990), provided the relevant ***peptides*** are processed and bound to MHC class I molecules. Other potential targets are breakpoint ***peptides*** of translocated oncogene products such as bcr/abl (Van Denderen et al., 1990). In viral systems, it has already been established that ***peptide*** ***vaccination*** for protective CTL induction is feasible (Aichele et al., 1989; Schulz et al., 1991; Kast et al., 1991). (46 Refs.)

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